Obesity and body mass index

Science, Biology

Obesity is known as having too much body fat. It can be defined by using a Body Mass Index, which is calculated using a person’s weight and height. A person who is considered obese will have a Body Mass Index of 30 kg/m2 or greater. A person with a BMI between 25kg/mg2- 29kg/mg2 is considered overweight. The excess adipose tissues gained in individuals with obesity can have many harmful effects on the body. Renner S. et al. tested the effects of obesity on the body. Previously, experiments that studied the pathophysiology of obesity were done using diet-induced rodents. In this study, they used the diet-induced model on Göttingen minipigs in order to interpret the triggers and mechanism of adipose tissue inflammation and the metabolic syndrome. Pigs were used in this study instead of rodents because their anatomy and physiology are more similar to humans. This similarity allowed the researchers to gain a better understanding of the triggers and the mechanisms leading to obesity, which can lead to better treatment for obesity and the diseases that are associated with it.

The study used twenty-six ovariectomized female mini-pigs ranging from eleven to thirteen months old and was performed over a 70-week period. The pigs were divided into two groups, lean and obese. The experimental group was the obese pigs, and the control group was the lean pigs. Both groups were fed a high fat/high energy diet. The variable in this study was the food portions. The food portions were gradually increased in the obese group in order for them to gain body weight. The portions of the lean group were calculated, so they met the energy and nutrient requirements necessary for an adult pig. Several measurements were taken during this study. The apparent digestibility of nutrients was calculated twice, once

during feeding week (FW) 20 and once during FW 70 (Renner, 2018). Body weight was also measured, and this was done two or three times per week. Blood samples were collected at the beginning of the study and during FWs 18, 30 and 54. In FWs 29 and 54, an intravenous glucose tolerance test was performed. A non-invasive heart rate measurement was also taken during FWs 59 and 69 (Renner, 2018). At the beginning of feeding week 60, eight of the obese mini pigs were chosen to be part of a chronic treatment trial.

They were divided into two groups. One group received a placebo which was 0. 9% sodium chloride while the other group received treatment with a GLP1R agonist. This treatment caused the pigs to lower their food intake. When the trial period was over the pigs were necropsied, and a biobank of organs, tissues and body fluids were collected. At the end of the study, the data between the lean and the obese group were analyzed. The apparent digestibility of nutrients was higher in the obese group compared to the lean group. At FW 70, the obese group’s body weight increased by 136% compared to the lean group (Renner et al., 2018). The heart rate of the obese pigs was significantly higher while resting and during activity. In the group that was part of the treatment trial, there was a decrease in body weight in those that received the GLP-1R agonist, while the control group continued to gain weight. The biobank samples that were collected showed a large amount of subcutaneous and visceral fat in the obese group, while it remained unchanged in the lean group (Renner et al., 2018). The obese pigs also developed inflammation in their adipose tissue, as well as, adipocyte necrosis. Glucose tolerance decreased in the obese pigs, and they developed insulin resistance. The pigs also experienced high plasma urea

concentration. Insulin resistance may have caused this since urea is a byproduct of gluconeogenesis (Renner, 2018).

One of the functions of insulin is to suppress gluconeogenesis, and with the organ not responding to it, it is unable to control the amount of glucose that the body produces. Insulin resistance is also seen in type-2 diabetes mellitus. Type-2 diabetes can be defined as insulin insensitivity which is caused by insulin resistance and a decline in insulin production caused by a malfunction of the beta cells (Olokoba et al., 2012). Insulin resistance in type-2 diabetes leads to an increase of fatty acids in the plasma. The increase of fatty acids lowers the amount of glucose that is transferred to the muscles (Al-Goblan et al., 2014). The transfer of glucose is also disrupted for the liver and fat cells (Olokoba et al., 2012). Al-Goblan et al., explain how as obesity increases, it releases substances such as non-esterified fatty acids (NEFA). The release of NEFA plays a role in the malfunction of beta cells. NEFA is usually necessary for insulin release, but if the concentration is increased, it impairs the synthesis of insulin and how insulin is released by the beta cells. (Al-Goblan et al., 2014). There has been an increase in the prevalence of obesity throughout the years, and it has become a significant public health problem. In 1999, it was estimated that 34 percent of U. S. adults were overweight and 27 percent were obese (Surgeon General, 2001). The rate of obesity is increasing in both developing and developed countries. It is estimated that the worldwide rate has increased to 10 percent and 15 percent in men and women, respectively (Renner, 2018).

There are many health risks associated with this disease. Studies have shown that the risk of death is increased by about 50 to 100 percent in people who are obese (Surgeon General, 2001). In addition to diabetes, obesity is associated with cardiovascular disease, hypertension, and cancers. Obesity has also become an economic problem. This disease has increased the cost of healthcare. There has been much money spent on preventing, diagnosing and treating it. There has also been much money lost because of this disease due to the loss of income by people who become disabled. In 2000, it was estimated that the cost of obesity was $117 billion (Surgeon General, 2001).

References:

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